

terial hypotension is the central factor in massive embolism and some form of vascular support is needed for survival. (Just-Viere, J. O., and Yeager, G. H.: *Massive Pulmonary Embolism, IV. The Value of Hyperbaric Oxygen as Therapy*, *J. Thor. Cardio. Surg.* 48: 185 (Aug.) 1964.)

**SPACE ATMOSPHERE** Airmen in a simulated space cabin were maintained at an average of 258 mm. of mercury total pressure with a  $P_{O_2}$  of 254 mm. of mercury and an average nitrogen partial pressure of 0.5 mm. of mercury. The atmosphere was well tolerated by the subjects who demonstrated very few of the previously described symptoms of oxygen toxicity. The aural atelectasis and nasal congestion were bothersome but did not interfere with mission completion. This was the only area where the presence or absence of nitrogen made a noticeable difference. The use of a single gas, 258 mm. of mercury oxygen, seems to be feasible for periods up to 30 days without any impairment of ability. (Herlocher, J. E., and others: *Physiologic Response to Increased Oxygen Partial Pressure*, *Aerospace Med.* 35: 613 (July) 1964.)

**EMPHYSEMA** Onset of cardiac failure or marked weight loss in patients with emphysema is associated with poor prognosis. Cardiac failure is the chief cause of death. Severity of dyspnea is not a useful prognostic sign. The degree of impairment either of the maximal voluntary volume, forced vital capacity, arterial carbon dioxide tension or arterial oxygen saturation is significantly related to prognosis. Impairment of only one of these parameters alone is a grave prognostic sign. (Boushy, S. F., and Coates, E. O.: *Prognostic Value of Pulmonary Function Tests in Emphysema*, *Amer. Rev. Resp. Dis.* 90: 553 (Oct.) 1964.)

**EMPHYSEMA** In subjects with emphysema vital capacity can be increased markedly if expiration is made at low flow rates rather than with maximal effort. The more severe the disease, the greater the increase in vital capacity. The same increase is not found in asthmatic subjects and is not found with increased oral pressure during expiration. (Schmidt, R. W.,

and others: *Effect of Air Flow and Oral Pressure on the Mechanics of Breathing in Patients with Asthma and Emphysema*, *Amer. Rev. Resp. Dis.* 90: 564 (Oct.) 1964.)

**DYSPNEA** Dyspnea was induced in 11 healthy volunteers by exposure to individual or combined stimuli including 7 per cent carbon dioxide, exercise and intravenously administered ethamivan. Stimuli which were ineffective in producing dyspnea individually became effective in combination. When dyspnea developed as a result of a combination of respiratory stimuli, withdrawal of a sufficiently potent stimulus resulted in the disappearance of the symptom. A relation of dyspnea to the net load of respiratory stimuli is suggested. A relation of dyspnea to the net load of respiratory stimuli is suggested. A rapidly increasing stimulus was more effective in producing dyspnea than one which achieved its maximum intensity gradually. (Kontos, H. A., and others: *Respiratory Stimuli in Dyspnea*, *Amer. J. Med.* 37: 374 (Sept.) 1964.)

**HYPERVENTILATION CONVULSIONS** After vigorous correction of severe hypercapnia by mechanical ventilation, generalized convulsions, multifocal central nervous system excitation, and coma may occur. The outstanding chemical abnormality in these patients is alkalosis with a striking elevation of bicarbonate concentration. Different organs might be expected to unload bicarbonate at different rates depending on their perfusion, anatomical nature of cell membranes, etc. Brain cells are particularly slow in their rate of unloading, and thus it is likely that the degree of brain intracellular alkalosis is even more severe than is apparent from the degree of extracellular alkalosis found in these patients. During ventilator treatment, arterial carbon dioxide tensions should be lowered gradually to avoid severe alkalosis. Existing alkalosis may be corrected by increasing arterial carbon dioxide tensions either by administration of carbon dioxide in oxygen or by deliberate hypoventilation with oxygen-enriched air. (Rotheram, E. C., and others: *Central Nervous System Disorder During Mechanical Ventilation in Chronic Pulmonary Disease*, *J.A.M.A.* 189: 993 (Sept. 28) 1964.)